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Terms	Documents
129 and @ad<19981118	15

Database: US Patents Full-Text Database; US Pre-Grant Publication Full-Text Database; JPO Abstracts Database; EPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

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Search History

Today's Date: 10/24/2001

DB Name	Query	Hit Count	Set Name
USPT	129 and @ad<19981118	15	L87
PGPB	173 with ((chromosome adj 2) or 2q31$3)	0	L86
PGPB	173 with golgi	0	L85
PGPB	173 with (nucle$2 or perinucle$2 or (perinucle$2))	5	L84
PGPB	173 with (intracell$4 near transport$2)	0	L83
PGPB	173 with rab$2	0	L82
PGPB	173 with (hypomethyl$5 or hypermethyl$5 or (hypo-methyl$5) or (hyper-methyl$5))	0	L81
PGPB	173 with (MCF7 or (MCF-7) or MCF7/$3 or mcf-7/$3)	0	L80
PGPB	173 with (MCF7 or (MCF-7) or MCF7/$3 or mcf-7/$3)	0	L79
PGPB	173 with ((multidrug or drug or (multi-drug)) adj resistan$2)	1	L78
PGPB	173 with (multidrug or drug or (multi-drug)) adj resistan$2	1	L77
PGPB	173 and 174	1	L76
PGPB	173 with 174	0	L75

PGPB	drug adj3 sensitiv$3	21	L74
PGPB	(guanine nucleotide binding protein$1) or ((G-protein$1) or (G adj protein$1))	108	L73
USPT	(guanine nucleotide binding protein$1) or ((G-protein$1) or (G adj protein$1))	3760	L72
JPAB,EPAB,DWPI	l68 and l39	0	L71
JPAB,EPAB,DWPI	l68 and l38	3	L70
JPAB,EPAB,DWPI	l68 and l41	0	L69
JPAB,EPAB,DWPI	(guanine nucleotide binding protein$1) or ((G-protein$1) or (G adj protein$1))	1250	L68
USPT	l65 same ((chromosome adj 2) or 2q31$3)	2	L67
USPT	l65 same (chromosome or 2q31$3)	57	L66
USPT	(guanine nucleotide binding protein$1) or ((G-protein$1) or (G adj protein$1))	3760	L65
USPT	l63 and (multidrug or drug or (multi-drug)) adj resistan$2	14	L64
USPT	l59 or l62	62	L63
USPT	l61 and @ad<19981118	62	L62
USPT	l48 and ((G-protein$1) or (G adj protein$1))	70	L61
USPT	l48 and ((G-protein$1) and (G adj protein$1))	10	L60
USPT	l58 and @ad<19981118	6	L59
USPT	l48 and (guanine nucleotide binding protein$1)	7	L58
USPT	l48 and (guanine nucleotide binding)	8	L57
USPT	l54 with chromosome	2	L56
USPT	l54 and (chromosome)	351	L55
USPT	estrogen receptor	1344	L54
USPT	l52 and @ad<19981118	30	L53
USPT	l4 and l51	33	L52
USPT	import$5 near nucle$2	353	L51
USPT	l49 and @ad<19981118	24	L50
USPT	l48 with l46	26	L49
USPT	intracell$4 near transport$2	437	L48
USPT	transport$2 with l46	2174	L47
USPT	nucle$2 or perinucle$2 or (perinucle$2)	143334	L46
JPAB,EPAB,DWPI	l41 and (intracellular adj transport$3)	0	L45
JPAB,EPAB,DWPI	l41 and transport$3	67	L44
JPAB,EPAB,DWPI	spgp or (s-pgp)	1	L43
JPAB,EPAB,DWPI	l39 and l41	1	L42
JPAB,EPAB,DWPI	(multidrug or drug or (multi-drug)) adj resistan$2	1832	L41
JPAB,EPAB,DWPI	l38 and l39	0	L40

JPAB,EPAB,DWPI	hypomethyl$5 or hypermethyl$5 or (hypo-methyl$5) or (hyper-methyl$5)	23	L39
JPAB,EPAB,DWPI	MCF7 or (MCF-7) or MCF7/$3 or mcf-7/$3	189	L38
JPAB,EPAB,DWPI	2q31$3	0	L37
JPAB,EPAB,DWPI	rab6c or (rab-6c) or (rab6-c) or (c-rab-6) or (c-rab6) or (crab-6) or crab6	0	L36
JPAB,EPAB,DWPI	wth3 or (wth-3)	1	L35
USPT	l8 and l29	0	L34
USPT	l30 and @ad<19981118	11	L33
USPT	l29 and l23	13	L32
USPT	l29 and l23	13	L31
USPT	l29 and l23	13	L30
USPT	2q31$3	18	L29
USPT	2q31	18	L28
USPT	l17 and l8	0	L27
USPT	l17 and ((multidrug or drug or (multi-drug)) adj resistan$2)	0	L26
USPT	l17 and (hypermethyl$5 or methyl$5 or (hyper-methyl$5))	0	L25
USPT	l17 and (hypermethyl$5 or methyl$5 or (hyper-methyl$5))	0	L24
USPT	hypermethyl$5 or methyl$5 or (hyper-methyl$5)	362666	L23
USPT	l17 and (tumor$1 or tumour$1 or cancer$1)	2	L22
USPT	l17 and perinucl$3	0	L21
USPT	l19 and perinucl$3	0	L20
USPT	l17 and golgi	0	L19
USPT	l17 and transport$3	0	L18
USPT	spgp or (s-pgp)	3	L17
USPT	l14 same l8	2	L16
USPT	l14 same l4	0	L15
USPT	hypermethyl$5 or (hyper-methyl$5)	81	L14
USPT	l12 and @ad<19981118	15	L13
USPT	l11 and l3	15	L12
USPT	l4 and l8	351	L11
USPT	l9 and l3	0	L10
USPT	l4 same l8	92	L9
USPT	MCF7 or (MCF-7) or MCF7/$3 or mcf-7/$3	1860	L8
USPT	l6 and @ad<19981118	40	L7
USPT	l4 and l3	41	L6
USPT	l3 same l4	0	L5
USPT	(multidrug or drug or (multi-drug)) adj resistan$2	5162	L4

USPT	hypomethyl$5 or hypermethyl$5 or (hypo-methyl$5) or (hyper-methyl$5)	157	L3
USPT	rab6c or (rab-6c) or (rab6-c) or (c-rab-6) or (c-rab6) or (crab-6) or crab6	0	L2
USPT	wth3 or (wth-3)	1	L1

STIC-ILL

From: Canella, Karen
Sent: Wednesday, October 24, 2001 11:02 PM
To: STIC-ILL
Subject: ill order 09/441,857

Art Unit 1642 Location 8E12(mail)

Telephone Number 308-8362

Application Number 09/441,857

1. Mutation Research, 1997 Apr, 386(2):153-161

2. FASEB Journal, 1997, Vol. 11, No. 9, p. A1201

STIC-ILL

0H301.F4
MC

From: Canella, Karen
Sent: Wednesday, October 24, 2001 11:02 PM
To: STIC-ILL
Subject: ill order 09/441,857

Art Unit 1642 Location 8E12(mail)

Telephone Number 308-8362

Application Number 09/441,857

1. Mutation Research, 1997 Apr, 386(2):153-161

2. FASEB Journal, 1997, Vol. 11, No. 9, p. A1201

Print Request Result(s)

Printer Name: cm1_8e12_gbelptr
Printer Location: cm1__8e12

- US005571687: Ok
- WO009941373A: Ok
- WO009731111A: Ok

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Copeman et al., "Linkage disequilibrium mapping of a type 1 diabetes susceptibilty gene(IDDM7) to chromosome 2q31-q33" Nature Genet., vol.9:80-85 (1995).

BSPR:

MRP possesses a nucleotide binding domain that is homologous with the ATP binding site of P-gp. See Marquardt, D., McCrone, S., and Center M. S., Cancer Res., 50: 1426, (1990). The mechanism(s) utilized by P190 to confer resistance to Adriamycin is not well understood but may involve the intracellular redistribution of Adriamycin away from the nucleus. See Marquardt, D. and Center, M. S., supra. Adriamycin is an inhibitor of topoisomerase II (Beck, W. T., Bull. Cancer, 77: 1131, (1990), which is an enzyme involved in DNA replication. Redistribution of Adriamycin away from the nucleus would therefore be an important component in cellular resistance to this drug. The studies published to date on P190 have utilized cell lines selected in vitro for resistance to Adrianycin (McGrath, T., Latoud, C., Arnold, S. T., Safa, A. R., Felsted, R. S., and Center, M. S., supra; Marquardt, D. and Center, M. S., supra; and Marquardt, D., McCrone, S., and Center M. S. Cancer Res., supra. The association of MRP (P190) with drug resistance was made by sodium dodecyl sulfate polyacrylamide gel electrophoresis (SDS-PAGE) of radioactive extracts prepared from Adriamycin-resistant HL60/Adr human leukemia cells labeled with 8-azido-alpha[.sup.32 P]ATP. See McGrath, T., Latoud, C., Arnold, S. T., Safa, A. R., Felsted, R. S., and Center, M. S., supra. The drug-resistance phenotype conferred by P190 is not limited to the anthracyclines. Epipodophyllotoxin resistance is linked to P190 expression. The IC.sub.50 s of HL60/S cells treated with Adriamycin and Etoposide were 0.011 .mu.g/ml and 0.39 .mu.g/ml respectively. The IC.sub.50 S for HL60/Adr cells (a HL60-derived cell line which is resistant to doxorubicin) treated with Adriamycin and Etoposide were 2.2 .mu.g/ml and >10 .mu.g/ml respectively. HL60/S and HL60/Adr cell lines do not express P-glycoprotein. HL60/Adr expresses P190. Thus, resistance to the anthracyclines and epipodophyllotoxins results from P190 expression.

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L51: Entry 21 of 24 File: USPT May 7, 1996

DOCUMENT-IDENTIFIER: US 5514600 A
TITLE: Mammalian guanine nucleotide binding protein with an ADP-ribosylation factor domain

APD:
19940927

BSPR:
ARFs are evolutionarily well conserved and present in all eukaryotes from Giardia to mammals (Kahn, et al. J. Biol. Chem. 263:8282-8287 (1988); Murtagh, et al. J. Biol. Chem. 267:9654-9662 (1992); Tsai, et al. J. Biol. Chem. 266: 8213-8219 (1991); Tsuchiya, et al. Biochemistry 28: 9668-9673 (1989); Tsuchiya, et al. J. Biol. Chem. 266: 2772-2777 (1991)). Immunologically, they have been localized to the Golgi apparatus of several types of cells (Stearns et al. Proc. Natl. Acad. Sci. (USA) 87:1238-1242 (1990)). ARFs are required for association of nonclathrin coat proteins with intracellular transport vesicles (Serafini, et al. Cell 67: 239-253 (1991)) and also appear to be critical during an early step in endocytosis as well as in nuclear vesicle fusion (Boman, et al. Nature (London) 358: 512-514 (1992); Lenhard, et al. J. Biol. Chem. 267:13047-13052 (1992)). GTP binding and hydrolysis may be involved in binding of ARF to membranes, and the nonhydrolyzable GTP analogue GTP.sub..gamma. S, but not GTP or GDP, promotes the association of cytosolic ARF with Golgi (Regazzi, et al. Biochem. J. 275:639-644 (1991)) 1991) or phospholipid membranes (Kahn, et al. J. Biol. Chem. 266:15595-15597 (1991); Walker, et al. J. Biol. Chem. 267: 3230-3235 (1992)).

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L34: Entry 1 of 11 File: USPT Mar 6, 2001

DOCUMENT-IDENTIFIER: US 6197525 B1
TITLE: Assay kits for detection and methods of inhibiting IL-17 binding

APD:
19980211

BSPR:
CTLA-8 refers to a cDNA cloned from an activated T cell hybridoma clone (Rouvier et al., J. Immunol. 150:5445; 1993). Northern blot analysis indicated that CTLA-8 transcription was very tissue specific. The CTLA-8 gene was found to map at chromosomal site 1a in mice, and at 2q31 in humans. Although a protein encoded by the CTLA-8 gene was never identified by Rouvier et al, the predicted amino acid sequence of CTLA-8 was found to be 57% homologous to the predicted amino acid sequence of an ORF present in Herpesvirus Saimiri, HVS13. The CTLA-8 protein is referred to herein as Interleukin-17 (IL-17).

BSPR:
Finally, one or more reversed-phase high performance liquid chromatography (RP-HPLC) steps employing hydrophobic RP-HPLC media, e.g., silica gel having pendant methyl or other aliphatic groups, can be employed to further purify a IL-17R composition. Some or all of the foregoing purification steps, in various combinations, can also be employed to provide a homogeneous recombinant protein.

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L34: Entry 3 of 11 File: USPT Feb 6, 2001

DOCUMENT-IDENTIFIER: US 6184031 B1
TITLE: DNA sequences that encode a natural resistance to infection with intracellular parasites

APD:
19960508

DEPR:
In the absence of either a known gene product or a reliable in vitro assay for gene function, a positional cloning approach to isolate the Bcg gene was chosen. Bcg maps on the proximal portion of mouse chromosome 1, close to the villin (Vil) gene (Malo et al., 1993, Genomics 16: 655-663). Using known marker loci and new anonymous probes obtained from a chromosome 1 micro-dissected library or generated by chromosome walking, a high resolution linkage map of mouse chromosome 1 was constructed (Malo et al., 1993, Genomics 16: 655-663). This mouse chromosome 1 segment of 30 cM overlapping Bcg, was found to be syntenic with a portion of human chromosome 2q in a segment delineated by loci COL3A1 (2q31-2q32.3) and COL6A3 (2q37) (Malo et al., 1993, Genomics 16: 655-663). After delineation of the maximal genetic and physical intervals defining the boundaries of the Bcg candidate gene region (Malo et al., 1993, Genomics 16: 655-663, and ibid 17: 667-675, respectively), a large segment of this domain was isolated in yeast artificial chromosomes (YAC), cosmid, and bacteriophage clones. This cloned genomic domain was analyzed for the presence of transcription units, and this eventually lead to the identification of a candidate gene for mouse Bcg.

DEPR:
Yeast chromosomes were prepared in agarose blocks. Agarose block slices (25 .mu.l) were incubated with restriction enzymes NotI, MluI, NruI, BssHII, SacII, or AscI (New England Biolabs, Beverley, Mass.) under the conditions recommended by the supplier. The digested DNA fragments were separated by electrophoresis in a 1% agarose gel (SeaKem/FMC.TM., Rockland, Me.) containing 0.5.times.TBE (1.times.TBE is 0.1 M Tris, 0.1 M boric acid, 0.2 mM Na.sub.2 EDTA [pH 8.0]) using a contour-clamped homogeneous electric field (CHEF-DRII.TM., Bio-Rad) configuration. Electrophoresis was performed at 200 V for 20 hr at 15.degree. C. with 15 s pulse times, allowing resolution of fragments in the range 20-400 kb. .lambda. oligomers (Pharmacia) and AB1380 yeast genomic DNA were used as size standards. Southern blots of these gels were prepared, and a physical map of the YAC DNA insert was determined after sequential hybridization of the blots to .sup.32 P-labeled individual single-copy probes from the region (Vil, D1Mcg101, D1Mcg102, D1Mcg103, D1Mcg104, and .lambda.Mm1C165), to plasmid fragments specific to each YAC cloning arm, and to total genomic mouse DNA. The left end probe was the larger and the right end probe the smaller of the two fragments produced by double-digestion of pBR322 with PvuII and BamHI. The restriction maps of the genomic DNA region and the corresponding YAC clones, together with the positions of the hybridization probes used for mapping, are shown in FIGS. 1B and 1C. The two YAC clones span a 400 kb segment and have a 170 kb region of overlap, which includes one of the entry probes, .lambda.Mm1C165 (FIG. 1C). A comparison of the composite restriction maps of the two overlapping YACs with that of genomic DNA shows concordance of both maps, suggesting that the two YAC clones carry non-chimeric inserts representative of the corresponding genomic DNA domain. Several additional rare-cutter sites were detected in both YACs that were absent in the genomic DNA (these sites are identified as closed symbols in FIG. 1C). The presence of newly accessible restriction enzyme sites in YAC

clones has been previously documented (Wilkes et al., 1991, Genomics 9: 90-95) and has been attributed to the absence of DNA methylation of cytosines in yeast cells.

L34: Entry 10 of 11 File: USPT Feb 16, 1999

DOCUMENT-IDENTIFIER: US 5871990 A
TITLE: UDP-N-acetyl-.alpha.-D-galactosamine: polypeptide N-acetylgalactosaminyltransferase, gAlnAc-T3

APD:
19960515

DEPR:
The GalNAc-T3 gene according to the present invention is a candidate gene for a recently identified insulin-dependent diabetes melitus susceptibility gene (IDDM7) localized to chromosome 2q31-33 (Copeman et al., 1995; Luo et al., 1995). The GalNAc-T3 gene is selectively expressed in pancreas, the target organ of diabetes type 1 autoimmunity, and co-localizes to chromosome 2q31. The GalNAc-T3 enzyme of the present invention was shown to exhibit O-glycosylation capacity beyond that of GalNAc-T1 and -T2, implying that the GalNAc-T3 gene is vital for correct/full O-glycosylation in vivo as well. A structural defect in the GalNAc-T3 gene leading to a deficient enzyme or completely defective enzyme would therefore expose a cell or an organism to protein/peptide sequences which were not covered by O-glycosylation as seen in cells or organisms with intact GalNAc-T3 gene. These findings strongly suggest that the GalNAc-T3 gene represents IDDM7. Described in Example 6 below is a method for scanning the ten coding exons for potential structural defects. Similar methods could be used for the characterization of defects in the non-coding region of the GalNAc-T3 gene including the promoter region.

DEPR:
The nucleic acids of the present invention may be flanked by natural human regulatory sequences, or may be associated with heterologous sequences, including promoters, enhancers, response elements, signal sequences, polyadenylation sequences, introns, 5'- and 3'- noncoding regions, and the like. The nucleic acids may also be modified by many means known in the art. Non-limiting examples of such modifications include methylation, "caps", substitution of one or more of the naturally occurring nucleotides with an analog, internucleotide modifications such as, for example, those with uncharged linkages (e.g., methyl phosphonates, phosphotriesters, phosphoroamidates, carbamates, etc.) and with charged linkages (e.g., phosphorothioates, phosphorodithioates, etc.). Nucleic acids may contain one or more additional covalently linked moieties, such as, for example, proteins (e.g., nucleases, toxins, antibodies, signal peptides, poly-L-lysine, etc.), intercalators (e.g., acridine, psoralen, etc.), chelators (e.g., metals, radioactive metals, iron, oxidative metals, etc.), and alkylators. The nucleic acid may be derivatized by formation of a methyl or ethyl phosphotriester or an alkyl phosphoramidate linkage. Furthermore, the nucleic acid sequences of the present invention may also be modified with a label capable of providing a detectable signal, either directly or indirectly. Exemplary labels include radioisotopes, fluorescent molecules, biotin, and the like.

ORPL:
Copeman, et al.,Nature Genetics, Linkage disequilibrium mapping of a type 1 diabetes susceptibility gene (IDDM7) to chromosome 2q31-q33, 9:80-85, 1995.

ORPL:

M. Fukuda, et al., "CRM1 is responsible for intracellular transport mediated by the nuclear export sgnal signal", Nature, 390:308-311, Nov. 1997.